David Taylor-Robinson
Brenda J. Thomas
Department of Genitourinary Medicine and Communicable Diseases
Imperial College School of Medicine
St. Mary's Hospital
London W2 1NY
United Kingdom

PII S0735-1097(00)00763-4

REFERENCES

1. Wong Y-k, Dawkins KD, Ward ME. Circulating *Chlamydia pneumoniae* DNA as a predictor of coronary artery disease. J Am Coll Cardiol 1999;34:1435–9.
2. Kaski JC, Camm AJ. *Chlamydia pneumoniae* infection and coronary artery disease. J Am Coll Cardiol 1999;34:1440–2.
3. Taylor-Robinson D, Thomas BJ. *Chlamydia pneumoniae* in arteries: the facts, their interpretation, and future studies. J Clin Pathol 1998;51: 793–7.
4. Boman J, Soderberg S, Forsberg J, et al. High prevalence of *Chlamydia pneumoniae* DNA in peripheral blood mononuclear cells in patients with cardiovascular disease and in middle-aged blood donors. J Infect Dis 1998;178:274–7.

REPLY

We thank Drs. Taylor-Robinson and Thomas for their interest in our study, which found circulating *Chlamydia pneumoniae (C. pneumoniae)* DNA to be associated with coronary artery disease (CAD) in men but not women (1). The association in men was moderate, with an odds ratio of 3.2 (95% confidence interval 1.1 to 8.9), and this was stronger than that reported in most serologic studies (2). We discussed possible reasons for the lack of an association in women, but because the number of male subjects was over twice that of females, the statistical power to detect a difference was strongest for men. In our study, patients and control subjects were defined according to the presence of CAD by angiographic criteria, an approach taken by other studies of *C. pneumoniae* and CAD (2), and, indeed, by studies that have investigated other coronary risk factors. We can justifiably claim that we have found an association between *C. pneumoniae* DNA and clinically significant atherosclerosis.

Our study and that of Boman et al. (3) are still the only published reports on circulating *C. pneumoniae* DNA and CAD, and we pointed out the differences in the reported prevalence of *C. pneumoniae* DNA. In our view, the finding that circulating *C. pneumoniae* DNA was found in 46% of a healthy, blood-donating population (3) is remarkable and an extraordinarily high level for any bacterium. However, although our study was far larger than Boman's, further work is required to clarify the situation, but we cannot comment on the unpublished data of the correspondents or their observations.

It would be generally accepted that evidence of current *C. pneumoniae* infection should be found before prescribing antibiotics to patients with CAD. At present, the presence of circulating *C. pneumoniae* DNA is the most accurate method of diagnosing current infection and is therefore a means of identifying suitable patients for intervention trials. This was also a view held by the correspondents (4), and we are therefore surprised by their statement that "fortunately," current antibiotic trials are being undertaken with complete disregard for the PBMC *C. pneumoniae* status. Fortunately, investigators running such trials do realize the potential importance of such a test (5).

Yuk-ki Wong
Wessex Cardiothoracic Unit
Southampton General Hospital
Tremona Road
Southampton, Hampshire S016 6YD
United Kingdom

Keith D. Dawkins
Michael E. Ward

PII S0735-1097(00)00764-6

REFERENCES

1. Wong Y, Dawkins KD, Ward ME. Circulating *Chlamydia pneumoniae* DNA as a predictor of coronary artery disease. J Am Coll Cardiol 1999;34:1435–9.
2. Wong YK, Gallagher PJ, Ward ME. *Chlamydia pneumoniae* and atherosclerosis. Heart 1999;81:232–8.
3. Boman J, Soderberg S, Forsberg J, et al. High prevalence of *Chlamydia pneumoniae* DNA in peripheral blood mononuclear cells in patients with cardiovascular disease and in middle-aged blood donors. J Infect Dis 1998;178:274–7.
4. Taylor-Robinson D, Thomas BJ. *Chlamydia pneumoniae* in arteries: the facts, their interpretation, and future studies. J Clin Pathol 1998;51: 793–7.
5. Grayston JT, Jackson LA, Kennedy WJ, et al. Secondary prevention trials for coronary artery disease with antibiotic treatment for *Chlamydia pneumoniae*: design issues. Am Heart J 1999;138:S545–9.

The Electric Cardiographic Abnormalities Are Not Hidden!

The report by Matetzky et al. (1) stimulates the following thoughts. There are two ways to interpret electrocardiograms (ECGs). One is to memorize patterns and the other is to use basic principles of electrocardiography, including the use of vector concepts, as described by Grant (2–6).

The tracing in Figure 1A of the report by Matetzky et al. reveals a left atrial abnormality; a mean spatial QRS vector that is directed at about +20° in the frontal plane (it changes direction during inspiration and expiration) and about 45° posteriorly; a mean spatial ST segment vector that is directed at +115° in the frontal plane and at least 90° posteriorly, indicating epicardial injury of the posterior wall of the left ventricle; a mean spatial T-wave vector that is directed at +90° in the frontal plane and 80° to 90° anteriorly, indicating posterior myocardial ischemia; and a large U wave in lead I.

The point is, the ST segment vector points toward an area of posterior epicardial injury. Furthermore, one can suspect that the ST segment vector is directed toward an obstruction in the circumflex coronary artery or its branches. It should be no surprise that the ST segment is elevated in leads $V_{7–9}$, because the transitional pathway was just beyond electrode position V_6. *The ST segment abnormality is not hidden.*

The follow-up discharge tracing in Figure 1B is also interesting. The left atrial abnormality has disappeared; an S_1, S_2 and S_3 conduction defect has developed; and the first half of the QRS complex is directed at about +40° in the frontal plane and markedly anterior, producing large abnormal R waves in leads V_1